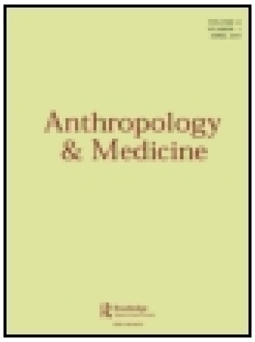

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Neoteny, psychiatric disorders and the social brain: hypotheses on heterochrony and the modularity of the mind

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ABSTRACT *The role of heterochrony in human evolution, defined as evolutionary change in the rate or timing of developmental processes, has been debated controversially among anthropologists. Early researchers suggested a single heterochronic shift towards neoteny, defined as the persistence of embryonic, foetal, or juvenile features of the ancestral species into adulthood of the descendants by slowing of growth and delayed maturation. Neoteny has been considered the hallmark of human evolution, leading to persistent curiosity, playfulness, and emotional attachment. Whereas neoteny-related non-specialisation was assumed to be of adaptive value to cope with different ecological environments, scientists now argue with reference to the thesis of a modular organisation of the mind that the complexity of the social environment forced the emergence of a highly specialised metacognitive capacity, referred to as the social brain hypothesis. This scientific shift also applies to hypotheses on the origin of psychiatric disorders, i.e. a failure of neoteny versus disturbances of social metacognition. This paper discusses the possible relation of heterochronic processes to Fodor's conception of horizontally and vertically organised modules of the mind in respect of its validity for the understanding of psychiatric disorders.*

Introduction

In the scientific history following Darwin's *The Descent of Man, and Selection in Relation to Sex* (1871), anthropologists have highlighted the relevance of divergent developmental processes for human evolution. In retrospect there has been a shift from emphasising the emergence of non-specialised adaptations in humans to different ecological environments (e.g. Lorenz, 1973; Montagu, 1962) towards focusing on highly specialised capacities, e.g. the emergence of metacognition, the latter presumed to have evolved due to an increasingly complex social environment (Brothers, 1990; Dunbar, 1998).

From an embryological perspective new physical characteristics of species

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may originate from an evolutionary change in the rate or timing of developmental processes, referred to as *heterochrony* (McKinney & McNamara, 1991). In the first decades of the last century the influence of embryology on evolutionary theory led to the idea that retaining juvenile characters into adulthood by slowing of growth (retardation) and delayed maturation, labelled as *neoteny* (derived from the Greek '*ne-teinein*': literally 'holding on to youth'; *Webster's New Encyclopedic Dictionary*, 1996) has played a decisive role in human evolution, in particular in respect of brain enlargement (Abbie, 1958; Bolk, 1926; De Beer, 1958; Keith, 1949; overview in Montagu, 1962, 1989; Gould, 1977). While primarily restricted to physical peculiarities in Bolk's original paper, his successors expanded the paradigm to cognitive, emotional and behavioural characteristics, namely, the persistence of curiosity, emotional attachment, and playfulness into adulthood (Montagu, 1962, 1989; Gould, 1977, 1981). These attributes were supposed to cause the uniquely human '*Weltoffenheit*' (cosmopolitanism; Gehlen, 1940; Lorenz, 1973). However, recent findings point out that the impact of heterochronic processes other than neoteny, i.e. *hypermorphosis* (defined as extension of growth periods, resulting in an 'overstepping' of the ancestral adult form by the addition of new structures and increase in size) have been under-recognised (Rice, 1997).

Yet, from an evolutionary perspective there is need for an explanation, why such an expensive organ like the human brain, weighing 2% of the body weight but consuming 20% of the energy intake, should have been favoured by selection (Aiello & Wheeler, 1995; Dunbar, 1998). Also, extending the juvenile period and delaying maturation must have had some advantages in human evolution, if one assumes that early maturation should maximise the overall reproductive fitness, whereas a delay would normally reduce fitness (Joffe, 1997). Evolutionary psychologists argue that the evolution of metacognitive capacities, that is, the ability to infer intentions and dispositions of other individuals 'mentalistically', referred to as theory of mind or so-called Machiavellian intelligence, has been crucial for maintaining complex social bands (Leslie, 1987; Whiten & Byrne, 1997). Building larger social groups, thereafter, may have been favoured by selection in hominid evolution. Thus, the increasing number of potential social encounters would require higher information processing capacities. Also, in respect of inclusive fitness theory the ability to manipulate con-specifics, e.g. by using tactical deception, would increase the individual's reproductive success (Whiten & Byrne, 1997). Extending the juvenile period may have been necessary to acquire the vast amount of possible social strategies (procedural rules), i.e. cooperation, tactical deception, exploiting the knowledge of other individuals, and *when to employ* the given strategies, referred to as meta-learning (Schmitt & Grammer, 1997; Whiten & Byrne, 1997).

In sum, tracing back the development of scientific conceptions of human evolution it becomes evident that the extension of juvenile developmental stages in humans lies at the interface between the hypotheses on neoteny and the emergence of the 'social brain'. However, these concepts have not been addressed with reference to heterochrony in general and the so-called modular organisation of the mind, respectively, the latter representing the renaissance of an influential paradigm in the cognitive sciences, in particular in theory of mind research (Baron-Cohen, 1995; Fodor, 1983).

Further, the same shift from non-specialisation to specialisation of human capacities applies to hypotheses on the origin of mental disorders. In the 1970s it has been suggested that some psychiatric disorders may be related to a failure of neoteny (Bemporad, 1991; Crow, 1995; Feierman, 1994; Jonas, 1974; Jonas & Jonas, 1974). However, the scientific evidence has been somewhat vague and largely speculative. On the other hand, there is growing interest in studying theory of mind abilities in psychiatric diseases with disturbances in autistic spectrum disorders being well documented (overview in Baron-Cohen, 1995).

In a nutshell, the heterochrony and the modularity hypotheses have not been converged in a heuristic sense. Therefore, the aim of this paper is to review the potential relation of heterochrony and social metacognition with reference to psychiatric disorders and the modularity of the mind.

Heterochrony and human evolution

The current literature lacks a generally accepted definition of heterochrony. For example, it is debated whether any change in shape or size represents heterochrony, whether the whole organism is unidirectionally involved, whether there is mosaic-like heterochrony in different tissues, and whether to apply the concept of heterochrony to either, growth curves or differentiation patterning (Deacon, 1990; Rice, 1997). Neoteny, as one of at least six forms of heterochrony, is defined as retaining embryonic, foetal, or juvenile features of the ancestral species into adulthood of the descendants by slowing of growth (retardation) and delayed maturation (De Beer, 1958; Gould, 1977; Montagu, 1962, 1989).

Historical notes

The phenomenon of neoteny was first described in amphibians by Karl von Baer in 1828. He discovered the development of mature germ cells in amphibian larval bodies. The term ‘neoteny’ was introduced by Swiss zoologist Julius Kollmann in 1884. Other expressions like ‘paedogenesis’ (von Baer), ‘fetalization’ (Bolk), ‘proterogenesis’ (Schindewolf), ‘bradygenesis’ (Ivanow), ‘pedomorphosis’ (Gould), and ‘juvenilization’ (Huxley) are more or less used synonymously. The term ‘neoteny’ embraces the others and is therefore the most comprehensive one (for an overview, see Montagu, 1962, 1989).

Morphology

In primate evolution, neoteny is most prominent in humans, but also present in our closest relatives, the great apes. For example, the pedomorphic skull form in the bonobo (*Pan paniscus*) compared to the common chimpanzee (*Pan troglodytes*) may be explained by changes in the rate and timing of similar patterns of development due to neoteny. It has been argued that this change may be related to reduced sexual dimorphism in morphology and behaviour (Shea, 1983; de Waal, 1996).

The obvious disparity between the large morphological and relatively small genetic distance separating humans and chimpanzees has led to the suggestion that relatively small changes of regulatory genes may account for the significant differences between these species, and that the process of neoteny has been crucial in hominid evolution, leading to a variety of physical peculiarities, the most important ones involving the skeleton, particularly the form of the skull, and brain enlargement (Gould, 1977; Montagu, 1962, 1989; Vrba, 1998) (Table I). Furthermore, some researchers have stressed the sexual dimorphism in humans with females being more neotenous than males as a result of sexual selection (Barber, 1995; Crow, 1995; Jones, 1995) (Table II). Analysing hominid skeletons, Groves (1989) found a shift towards neotenous features from *Australopithecus* through *Homo sapiens*. However, studying cross-sectional growth curves of juvenile and adult human crania of the gorilla (*Gorilla gorilla*), the common chimpanzee (*Pan troglodytes*) and the orangutan (*Pongo pygmaeus*), Dean and Wood (1984) did not verify that unidirectional modifications of the

TABLE I. Neoteny-related physical characteristics in man (proposed by Bolk, 1926; Gould, 1977; Montagu, 1962, 1989).

Neotenous features of the skull	Other neotenous features of the skeleton	Neotenous features of the integument	Other neotenous features
Retention of cranial flexure	Small flat face (orthognathy)	Full lips	Persistence of labia majora and hymen
Forward position of foramen magnum	Small teeth, late eruption	Relative hairlessness of body	Anterior position of the vagina
Retarded closure of cranial sutures	Small jaws	Thin nails	Persistence of penile prepuce
Absence of heavy brow ridges and cranial crests	Non-rotation of big toe	Lack of pigment in some groups	Large eyes
Roundheadedness (brachycephaly)	Curvature of pelvis axis		Large volume of brain
Orbits under cranial cavity			Low birth weight
Large braincase			Prolonged growth period
High ratio of neurocranium to lower face			

TABLE II. Physical neotenous features in females compared to males due to sexual selection (after Barber, 1995; Jones, 1995; Montagu, 1989).

Larger eyes in relation to face size
Smaller nose
Higher cheek bones
Smaller chin
Thinner jaws
More pronounced hairlessness of the skin
Relative lack of pigmentation in skin and hair
Smaller feet
Deposition of fat on the distal extremities
Shorter stature

timing or pattern of growth of the cranial base of a common ancestor sufficiently explain the observed morphological differences between these species (see also Godfrey & Sutherland, 1996). Rather, human growth curves may best be explained as a combination of neoteny and sequential hypermorphosis (McKinney & McNamara, 1991; Rice, 1997). Moreover, some aspects of the neoteny hypothesis concerning the development of the human brain have been criticised: the human brain ceases growing later in humans than in apes, but during early ontogeny, it grows rapidly, thus, there is no overall slowing of growth (Godfrey & Sutherland, 1996). The degree of cortical folding is associated with brain size only for the first part of brain growth. Human cortical proportions evolved when the brain enlarged in size. Differences of areas whose sulci differ in length between the two hemispheres but having similar degrees of convolutedness reveal that the sizes of gyri are asymmetric in the two hemispheres, suggesting that the placement and orientation of sulci requires other than simple allometric explanations, e.g. gyrogenesis, and cannot merely be explained as a function of neoteny (Armstrong *et al.*, 1995). According to Deacon's (1990) analysis the human brain grows as if it was in a giant ape body, at the same time regulating body growth as if it were the size of a chimpanzee brain, mediated by a 'decoupling' of growth in different brain regions which is not consistent with a simple allometric shift. However, the human brain continues growing into the third decade of life, with ongoing myelinisation particularly of the association cortex until adulthood. Thus, there is ample evidence that heterochronic processes have been crucial in human evolution, with neoteny and hypermorphosis being the most influential ones.

Behavioural neoteny

Lorenz (1953, 1973), Montagu (1962, 1989), Gould (1977, 1981), and others have proposed that neoteny also accounts for a variety of cognitive, behavioural and emotional features unique to humans, emphasising explorative behaviour as

TABLE III. Proposed characteristics due to 'behavioural neoteny' (after Gould, 1977; Lorenz, 1973; Montagu, 1962, 1989;).

Persisting learning abilities
Curiosity
Play
Flexibility
Explorative behaviour
Behavioural adaptability
Prolonged dependency period
Persistent emotional attachment

the key feature of persistent brain plasticity (Table III). Abandoning instinctive patterns of behaviour, for example, may have reinforced behavioural flexibility. Escaping from specialisation may have allowed for coping with different ecological environments (Lorenz, 1953). Furthermore, it has been proposed that neoteny plays a major role for retaining strong emotional attachment into adulthood arising from the extremely expanded time of dependency in infancy (Montagu, 1989). More recently, Bjorklund (1997) has argued that immature sensory systems may protect from overstimulation during infancy and childhood, and poor metacognition may provide the opportunity to learn by imitation without recognising the inadequacy of the attempts and may benefit development of social strategies in the long term, therefore, being adaptive to cope with the demands of the social environment during early ontogenetic stages (Bjorklund, 1997). However, the line of arguments appears to confuse cause and effect.

Sexual selection

Sexual selection has lead to a mild sexual dimorphism in humans concerning neotenous characters with females being more neotenous than males (Barber, 1995; Jones, 1995). These differences are partly probably mediated by the action of sex hormones. It has been argued that in males testosterone-dependent secondary sexual characteristics may signal immunological competence, whereas oestrogen-dependent, relatively more neotenous features of females may correlate with health and reproductive fitness and may therefore be found attractive (Barber, 1995; Jones, 1995). Perrett *et al.* (1998) studied facial attractiveness by asking subjects to choose the most attractive faces from continua that enhanced or diminished differences between the average shape of female and male faces. While feminised to average shapes of female faces were preferred by males, the results for male faces were more ambiguous. Masculinised facial characteristics increased both, perceived dominance and negative attributions to paternal investment, the latter limiting sexual dimorphism while favouring neoteny in humans. Moreover, it has been argued that neoteny-related facial characteristics may have an anti-aggressive effect (Jones, 1995). Yet, to date it is ambiguous whether this may be related to the '*Kindchenschema*' ('childlikeness'), which is doubtless a phylogenetically old trait (Lorenz, 1978). Coss and Schowengerdt (1998) found that pedomorphic female facial profiles were found vulnerable and youthful by both, males and females. However, in this study slightly archaic profiles were rated more voluptuous and cuter, compared to the most pedomorphic profile, suggesting the persistence of an 'ancestral-assessment system' favouring archaic features (Coss & Schowengerdt, 1998).

In addition, Crow (1993, 1995) suggests that a genetic change on a X–Y homologous locus of the sex chromosomes controlling hemispheric asymmetry may have become a target of sexual selection and that neoteny has been involved in subsequent progressive increase of brain size.

Hypotheses on the relation of neoteny to psychiatric disorders

Schizophrenia

As early as in 1871, Ewald Hecker, probably unaware of evolutionary ideas, stated in his essay on hebephrenia that the disorder is characterised by a lack of maturation, indicated by a pathologically permanent state of puberty. With respect to the displayed manneristic, exaggerated behaviours, Hecker concluded that the "affected individuals have slowed down early in life in their physical and emotional development" (translation quoted from Sedler, 1985).

More recently, referring to clinical observation, Bemporad (1991) has hypothesised that the lack of curiosity, motivation and playfulness, and the impaired capacity to learn in patients with 'dementia praecox' may indicate a 'failure of neoteny' as a result of dysfunction of regulatory genes to program structural genes to produce enzymes necessary for neoteny. This may lead to premature psychological ageing in these individuals (thus, in some respect, holding the opposite position compared to Hecker). Negative symptoms, for example, affective blunting, avolition apathy, and anhedonia may reflect a loss of cognitive, emotional, and behavioural neotenous attributes, whereas positive symptoms like delusions and hallucinations may be viewed as attempts to compensate for neuronal understimulation. Chronic schizophrenia, Bemporad deduced, may be loosely compared to the life course of 'subprimate mammals' (Bemporad, 1991).

In addition, Crow (1995) proposes a failure of neoteny corresponding to a lack of cerebral asymmetry in psychotic disorders (see above). In schizophrenia, for example, arrested development of normal cerebral asymmetry is well documented (Crow, 1990, 1991). After Crow, psychosis arises at the extremity of a distribution of variation in cerebral structure generated in the course of hominid evolution. These general aspects of the origin of psychoses may also account for other severe developmental disorders, e.g. autism and Asperger's syndrome (Crow, 1995). Recent findings suggest that cerebral asymmetry and the predisposition to psychosis may be linked to a defective gene regulating the relative growth of the two hemispheres, located on an X–Y homologous region of the sex chromosomes (Crow, 1993; Laval *et al.*, 1998). Variation of gene expression thereafter may also account for the relatively stable incidence rates in different populations (Crow, 1995).

In his etiological speculation about schizophrenia, being the consequence of a partially nocturnal brain, Feierman (1994) discusses the possible impact of neoteny. Admitting the weakness of the presented hypothesis, selection pressure for neoteny in human evolution could have carried along the phylogenetically older trait 'nocturnalism'. However, if so, persons suffering from schizophrenia should express more physical neotenous traits than average which is obviously not true (Feierman, 1994). Therefore, a link between schizophrenia and neoteny appears quite unlikely.

Neuroses and personality disorders

Classical psychoanalytic theory poses an obstructed development of personality in neurotic disorders, leading to incomplete maturation, and persistence of infantile behaviours (Freud, 1938). Jonas and Jonas (1974) relate neurotic patterns of behaviour and psychosomatic symptoms to neoteny by inferring the retention of 'archaic' responses. These authors argue that man has retained phylogenetically old responses that may recur in conflicting situations, e.g. submission rituals, mock death, disgorging of food orally or evacuating it rectally, the diving reflex, hoarding, hibernation, self-elimination (suicide), etc., due to his neoteny-related unspecialised morphology, yet confusing neoteny with recapitulation (Montagu, 1989).

Some researchers and popular writers infer that the biological process of neoteny may have been favoured by cultural evolution, indicated by a strong tendency in humans to keep neotenous traits as long as possible, even in the elderly (e.g. resulting in a growing fitness and beauty industry, and striving for juvenile appearance and behaviour in general, at least in Western cultures; Novak, 1982, quoted from Montagu, 1989; Bly, 1996). Hence, it has been speculated that interference of cultural and biological evolution may facilitate the persistence of immature behaviours with possible impact on normal psychological maturation, therefore leading to an increase of neuroticisms. However, cross-cultural research is lacking and the available accounts are anecdotal.

The modular organisation of social metacognition

According to Fodor's (1983) original conception, brain functions are organised in more or less independently operating 'vertical' modules. Modules are domain-specific, operate quickly, have a characteristic ontogenetic course and pattern of breakdown, and a distinct cerebral representation. In contrast to the 'general intelligence hypothesis' the modular view proposes multiple 'intelligences' that are specifically designed to solve problems of adaptive significance

(Brothers, 1990). Social metacognition thereafter is represented by an innate, domain-specific, hierarchically organised module that evolved due to natural selection pressures of the social environment.

Metacognitive capacities are particularly important for social interactions that rely on mutual cooperation. According to Cosmides' (1989) 'social contract theory', a social contract relates "*perceived benefits* to *perceived costs*, expressing an exchange in which an individual is required to pay a cost to an individual (or group) in order to be eligible to receive a benefit from that individual (or group). Cheating is the failure to pay a cost to which one has obligated oneself by accepting a benefit, and without which the other person would not have agreed to provide the benefit" (Cosmides, 1989). Cooperation ('reciprocal altruism'; Trivers, 1971) between two or more individuals could therefore not have evolved unless cheater detection became possible, i.e. by developing the capacity to infer other persons' mental states and to discriminate the true mental state from overt 'false' behaviour. However, the facilitating algorithms of social metacognition do not work optimally in real life situations, because optimal working is time consuming and complex. Usually, decisions about social matters must be made quickly. Thus, due to simplification and time limitation social reasoning processes bare the risk to err (Gigerenzer, 1997). Further, social reasoning is influenced by emotional states and their corresponding body states. This may have forced the evolution of brain structures involved in perception of body states and emotions as well as their subjective correlates we call 'feelings' (Damasio, 1995, 1996).

Metacognitive capacities are acquired following distinct ontogenic stages, i.e. understanding that seeing leads to knowing, perspective taking, and ultimately comprehending several levels of 'intensionality' (knowing that someone else knows that someone else knows something and so on). Not until the age of three to four years is the child able to distinguish between his or her own and others' beliefs, for example, that someone may hold false beliefs, referred to as first-order false belief (Baron-Cohen, 1995). At the age of six to seven years, children learn to understand second-order representations, i.e. knowing that someone else knows that someone else knows something, which is, for example, necessary to distinguish jokes from lies (Winner *et al.*, 1998). A *faux pas* may not be reliably understood before the age of 9–11 years, with girls performing well on corresponding tasks earlier than boys. A *faux pas* happens when someone says something he or she should not have said, not grasping his or her mistake. Understanding this requires a developmentally advanced theory of mind because simultaneous representation of two mental states is obligatory: that of the person who commits the *faux pas*, and that of the second person involved who may feel hurt or irritated (Stone *et al.*, 1998; overview in Brüne, in press).

Proposed brain structures involved in social metacognition

For the scope of this paper it is useful to outline shortly the neurophysiological basis of social cognition, because it may be hypothesised that the brain regions involved correspond largely with the modifications that took place due to heterochrony.

Recently, Damasio (1996) has proposed a neural circuit for social metacognition including the following candidate brain regions: the frontal lobes comprising the working memory in the dorsolateral area, the medial prefrontal cortex involved in the subjective emotional judgement of social interactions, the temporal lobes, the anterior cingulate cortex, the amygdala and the posterior association cortex, and their interconnections (Damasio, 1995). It is of importance that these changes not only occurred due to brain enlargement but also due to a greater increase of density of neurons particularly in the limbic system than expected from allometric analyses (Ploog, 1988).

The evolution of language following specialisation of the hemispheres is probably the most impressive feature in humans. There is evidence for a disparity of cognitive capacities between the sexes with females being superior in verbal skills compared to males, whereas males perform better on spatial abilities, suggesting a relation to sexual selection (Crow *et al.*, 1998). Yet, the relation to a single heterochronic process, i.e. neoteny, is at best speculative.

Disturbances of social metacognition

Autism

There is a vast amount of literature on deficits of social cognition in autism, which is impossible to refer to in detail here (overview in Baron-Cohen, 1995). In essence, autistic children are severely impaired in establishing social relationships. They do neither seek eye or bodily contact, nor show expressive gestures, indicating marked disturbances of normal affective bonding. Autistics engage frequently in stereotyped behaviours, possibly resulting from sensory understimulation (Ridley & Baker, 1982; Brothers, 1990). They are unable to understanding the emotional content of facial expression. As an outstanding feature, persons with autism have much greater difficulties in inferring mental states of other individuals compared to mentally retarded children. There is substantial evidence that autistics do not develop a sufficient theory of mind at all. For example, they fail in comprehending second-order false beliefs compared to age- and IQ-matched controls (Baron-Cohen, 1995). This has lead to the assumption that autistic disorders represent an inborn absence of social cognition, whereas other domains are not necessarily affected (Brothers, 1990; Baron-Cohen, 1995). These findings have been replicated by numerous clinical experiments using picture stories and other especially designed tests for comprehension of perspective taking (overview in Baron-Cohen, 1995).

Schizophrenia

Persistent misinterpretation of intentions of other persons is an outstanding feature of the paranoid psychoses (Brothers, 1990). Characteristically, the behaviour of other persons is pathologically related towards the self, primarily by exaggerated inference of tactical deception, thus leading to persecutory delusions or delusions of being influenced by others. Inferences of benevolent attitudes, e.g. cooperative or erotic intentions, are much more infrequent. Recently, systematic studies have focused on the investigation of false beliefs in psychotic disorders testing theory of mind skills and causal attributions by using short descriptions of social interactions and specifically designed attributional questionnaires (Bodner & Mikulincer, 1997; Doody *et al.*, 1998; Drury *et al.*, 1998; Frith & Corcoran, 1996; Kinderman & Bentall, 1997).

Patients with schizophrenia thereafter perform significantly worse on false belief tasks than non-schizophrenic controls, e.g. patients with affective disorders, mild learning disabilities, and normal controls (Frith & Corcoran, 1996; Doody *et al.*, 1998; Drury *et al.*, 1998). However, these differences are restricted to acute episodes, while after recovery there are no significant differences between the groups (Drury *et al.*, 1998). Patients with delusions of persecution and reference do not differ from the control groups at all, with the exception of poorer performance on memory tasks as a measure of comprehension of the applied test stories (Drury *et al.*, 1998). Consistent with the assumption that the ability to understand false belief tasks may deteriorate with chronicity of the psychotic disorder, performance on theory of mind tests may be significantly influenced by IQ, attention, concentration and selective memory impairments (Frith & Corcoran, 1996; Doody *et al.*, 1998; Drury *et al.*, 1998).

Comparing causal attributions using an attributional questionnaire, paranoid patients tend to blame other individuals for hypothetical negative social events, whereas depressed patients preferentially choose self-blaming causes. Healthy controls tend to choose external causes that do not involve other persons but rather circumstantial attributions (Kinderman & Bentall, 1997). In addition, depressive and paranoid-like responses in healthy subjects after failure in unsolvable problems depend on the focus of attention. When focusing attention on the self, personal failures produce depressive-like reactions. In contrast, when the threatening agent (the experimenter) is the focus of attention, subjects tend to react with paranoid-like reactions to personal failures (Bodner & Mikulincer, 1998).

Discussion

The extreme extension of the juvenile period is an outstanding feature of human evolution. However, rather than being restricted to neotenous modifications, growth allometries in humans are probably much more complex with sequential hypermorphosis being likely involved (McKinney & McNamara, 1991; Rice, 1997). Heterochronic processes have generally been under-recognised in medicine, for example, neoteny-related morphological changes may also account for difficulties in allometric approach for interspecies scaling in pharmacology, to date widely neglected in psychopharmacological research (Ritschel *et al.*, 1992; Yates & Kugler, 1986).

Evolutionary pressures towards the extension of the juvenile period root at least in part in the development and refinement of social interaction skills, forced by increasing social complexity (Dunbar, 1998; Joffe, 1997). Further, the shift towards neotenous traits may have been amplified by sexual selection and the need to reduce intraspecies aggression (Jones, 1995).

Both, the neoteny hypothesis and the social brain hypothesis have been independently related to psychiatric disorders. While this may be fruitful, since pathology contributes much for the understanding of physiology and vice versa, the question has not been addressed whether these hypotheses may be intertwined. It is obvious that the extension of juvenile stages in humans lies at the interface between these at first sight conflicting conceptions.

Neoteny in human evolution has lead to a variety of physical peculiarities, with brain development being the most controversially discussed one (Table I). In fact, it is debatable whether brain evolution is linked to one or several heterochronic conditions or, rather, being the result of complex reorganisation and differentiation processes that do not represent simple allometric shifts (Armstrong *et al.*, 1995; Deacon, 1990; Rice, 1997). In addition, it is speculative to associate certain cognitive, behavioural, and emotional peculiarities to morphological characters. However, neoteny has been labelled the 'hallmark' of human evolution since curiosity, play, persistent educability and emotional attachment have been associated with neoteny (Montagu, 1962, 1989; Gould, 1977). In the reverse, disturbances of these features have given rise to the hypothesis that there might be a failure of neoteny in psychiatric disorders. In fact, there is a (superficial) resemblance between a deficit of the cognitive, behavioural and emotional characters ascribed to neoteny, and negative symptoms in schizophrenia, respectively. Yet, there are some objections to this hypothesis. First, if schizophrenia is related to a failure of neoteny, is 'behavioural neoteny' independently inherited from physical neoteny? To my knowledge, there are no marked physical deviations in schizophrenia that hint towards a general failure of neoteny including morphological features. Also, it is not plausible that brain asymmetry is simply a result of neoteny. Clinically, it is also unlikely that a pure failure of neoteny may account for schizophrenia, since beside negative symptoms 'exaggerated' neotenous behaviours, e.g. mannerisms, may occur simultaneously. It appears also at best conjectural to compare schizophrenia to a subprimate condition, and to relate neoteny to nocturnalism, respectively. The role of the proposed defective regulatory genes that may account for schizophrenic brain abnormalities requires scientific confirmation. Although empirical findings suggest that single gene inheritance of brain asymmetry due to sexual selection may account for the susceptibility for psychotic disorders the association with neoteny is vague (Crow, 1995; Laval *et al.*, 1998). For example, in Down's syndrome there are exaggerated physical neotenous characteristics, beside vestigial and atavistic features, suggesting a gene locus on Chromosome 21 involved in these variations (Opitz & Gilbert-Barness, 1990).

Second, the hypotheses on the relation between neurotic and personality disorders to neoteny confuse neoteny with the recapitulation hypothesis or biogenetic rule. In human ontogenetic development there is at least no evidence for the recapitulation of phylogenetically older behavioural traits (Medicus, 1992). As De Beer (1958) puts it, from an embryological viewpoint phylogeny is the result of ontogeny rather than being its cause. Whether cultural evolution influences the biological process of neoteny, as supposed recently by popular writers (Bly, 1996), requires further transcultural research. Persistent or reactivated immature behaviours in neuroses and personality disorders do not necessarily involve neotenous developmental disturbances. Rather, slowing of maturation and prolonged childhood are important for normal development, particularly for the acquisition of procedural rules of social interactions (Bjorklund, 1997; Joffe, 1997; Schmitt & Grammer, 1997).

Consistent with the vulnerability concept of psychotic disorders (Weinberger, 1986; Walker *et al.*, 1999) neoteny-related persistent brain plasticity and delayed maturation, on the other hand, may enhance the susceptibility to physical and functional disturbances, regardless whether environmentally, infectious, or socially triggered (Crow, 1991). Therefore, psychiatric disorders may be the 'price' for the delay of maturation and for the rapid increase of brain size during the past 150,000 years. For example, normal development of the human nervous system is particularly dependent on sensory input. Social deprivation leads to stereotyped and self-injurious behaviour, resulting from an alteration of dopamine receptor sensitivity (Ridley & Baker, 1982). The assumption of an enhanced susceptibility to psychotic disorders by delayed maturation may be underlined by the fact that most psychiatric disorders originate in early childhood, when the growth rate of the brain and acquisition of learned material is particularly high. Clinical manifestation of psychiatric disorders (which is different from their origin) is variable throughout the entire human life span with some predisposing stages, e.g. early childhood, puberty, adolescence, and menopause, suggesting that hormonal trigger-effects are involved in regulating the neotenous development, as already assumed by Bolk (1926). While human neoteny represents a generalised rather than a specialised condition, allowing to cope with a wide range of ecological environments by abandoning restricted (instinctive) patterns of behaviour, the social brain, in this regard, represents the reverse condition, being a highly specialised evolutionary 'invention'. Thus, the development of the social brain can clearly not be explained as neotenous capacity. A comparison of theory of mind skills of the great apes to humans does not suggest that such a 'costly' development may have appeared only in the juvenile stages of a human ancestral species, disappearing during the subsequent course of ontogeny. If adhering to the paradigm of the association between heterochrony and behaviour, this argumentation gave rise to speculation whether the development of a social brain may partly be regarded as a function of sequential hypermorphosis, thus, if we may speak of 'behavioural hypermorphosis' analogous to 'behavioural neoteny'. By definition, in hypermorphosis the adult condition of the ancestor is present in unreduced form in the development of the descendant (De Beer, 1958). However, while no longer representing the terminal stage, hypermorphosis provides the possibility to add new characters.

Thus, the acquisition of advanced steps of social intelligence, e.g. additional levels of intensionality and perspective taking may be considered hypermorphotic, when comparing theory of mind abilities in the great apes to the ontogenetic acquisition during human infancy and childhood. Although largely speculative in light of the present data, if higher mental functions are associated with different heterochronic processes, it may be reasonable to speak of 'behavioural heterochrony'. Classic ethology, for example, has revealed how behaviours, e.g. fixed action patterns, are linked to anatomical structures. For example, for the efficacy of any mating display in birds, the presence of releasing signals, mediated by physical characters, e.g. feathers, colours, etc., is indispensable. Correspondingly, explorative behaviour, learning capacities, and emotional attachment may be linked to neoteny, and theory of mind abilities to hypermorphosis, respectively. Although hypothetical, these assumptions are principally testable, for example, by correlating allometric growth curves of the great apes and humans with behavioural characters, as well as studying correlations of behavioural traits and heterochronic features outside primate evolution.

From the present scientific data, it appears more efficacious to focus on the analysis of the modularity of the mind. The concept of modular functioning of the mind has been quite influential in the cognitive sciences (Fodor, 1983). Gigerenzer (1997) distinguishes domain-general from domain-specific modules. Modules are hierarchically organised, triggered by algorithms that allow quick decisions. The efficiency of social algorithms, however, is constrained by limited time and knowledge, because decisions concerning social interactions have to be carried out instantaneously. Thus, these algorithms do not operate optimal, but rather 'satisficing', combining cognitive, emotional and motivational processes (Gigerenzer, 1997). Taking into account possible disturbances during acquisition of procedural and meta-rules (Schmitt & Grammer, 1997), there are several potential modes of disturbances of social cognition in psychiatric disorders. In Fodor's (1983) terms explorative behaviour, persistent learning abilities, and emotional bonding (regardless whether related to neoteny or not) may be considered as domain-general 'horizontal' faculties that may be compulsory for the subsequent development of specialised 'vertical' modules. In the probably heterogenous disorders summarised as schizophrenia, domain-general impairments of cognitive abilities, curiosity, and emotional attachment predominate at first sight. This may lead to dysfunctional social meta-learning, i.e. how and when to apply social behavioural strategies. In schizophrenia, functionally disturbed triggering and 'satisficing' algorithms may lead to abnormal inference of tactical deception in other individuals. Studies on healthy subjects reveal that under specific circumstances involving the attentional focus and motivations depressive-like and paranoid-like responses may emerge in any individual, suggesting a relative instability of appropriate application of social skills (Bodner & Mikulincer, 1998). However, in normal ageing there is no evidence of a decline of theory of mind abilities. Rather, in the study of Happé *et al.* (1998) older participants perform even better on theory of mind tasks than younger individuals, suggesting a process of 'modularisation' throughout the life span.

However, whether there is a decline of theory of mind skills in dementia has not been systematically investigated yet.

Functional or physical damages of the candidate brain structures of social metacognition are particularly involved in psychiatric disorders. For example, there is a vast amount of literature on 'hypofrontality' in chronic schizophrenia, conduct disorders and others. The relation of the proposed brain structures responsible for social metacognition and heterochronic processes is at best hypothetical. It may be speculated that these brain regions correspond to those, altered by neoteny and sequential hypermorphosis, i.e. the association cortex, but the role of mosaic heterochrony, gyrogenesis and other reorganisation processes is ambiguous (Armstrong *et al.*, 1995; Deacon, 1990).

In summary, evolutionary pressures towards an extension of the juvenile period may have forced a developmental shift towards physical and possibly behavioural neoteny and sequential hypermorphosis. Further research may clarify whether these non-specialised and highly specialised conditions have modular equivalents, representing domain-general and domain-specific faculties of the mind. However, these modifications in human evolution may exemplify how natural and sexual selection operate on proximate mechanisms and how these changes are ultimately realised. The coexistence of extremely unspecialised (neotenous) and highly specialised (hypermorphotic) characteristics in humans may be a unique condition in primates. Yet, to date the consequences are obscure. Evolutionarily spoken, non-specialised species normally have a greater likelihood of survival than specialists. In respect of humankind we will unfortunately never know which way *Homo sapiens* will take.

Giving a complete overview on social brain, neoteny and psychiatric disorders is virtually impossible and well beyond the scope of this article. Thus, flaws and shortcomings are likely to be inevitable. However, 'thinking modular' may contribute to a better understanding of how cognition, emotion, and behaviour are interrelated and how disturbances of different modules affect the clinical pictures of psychiatric disorders. In addition, it may possibly provide new insights useful for therapeutical work.

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